Chronic exertional leg pain: discrete syndromes or a continuum?

Phil Newman looks at this common issue and the difficulties practitioners have in making a diagnosis.

Shin pain is a common complaint presenting to sports physiotherapists. Up to 82 per cent of runners have been found to experience exercise-related shin pain over a single running season (Reinking et al 2010). Chronic exertional leg pain (CELP) or exercise-related leg pain are common terms within the current literature, encompassing a group of diagnoses familiar to clinicians: medial tibial stress syndrome (MTSS), chronic exertional compartment syndrome (CECS), tibial stress fracture, and popliteal artery entrapment syndrome (PAES). One of the difficulties in interpreting the literature in the area of CELP is that these diagnostic entities are not well differentiated in study samples and co-existence of each condition is common. Advances in imaging techniques and other technologies are helping to grow our understanding of these various conditions and their inter-relationships.

MTSS: 16–35 per cent of runners

The typical symptoms of MTSS are '…pain along the posteromedial border of the tibia that occurs during exercise, excluding pain from ischaemic origin or signs of stress fracture' (Yates & White 2004). Pain extends over at least a 5 cm length of the distal one third of the tibia. MTSS has been suggested to be a combination of myofascial strain, enthesiopathy, periostitis and bone stress.

Histological studies (Bouche & Johnson 2007) and cadaveric studies have not conclusively determined myofascial strain to be a component (Mubarak et al 1982, Michael & Holder 1985, Bhatt et al 2000, Johnell et al 1982, Stickley et al 2009, Beck & Osternig 1994, Saxena et al 1990). The portion of the tibia where symptoms occur (distal

one third) is most often absent of myofascial attachments making myofascial traction induced mechanisms unlikely (Stickley et al 2009, Beck & Osternig 1994).

However, pain in the posteromedial musculature is a consistent clinical feature of MTSS. It is more likely that this muscular tenderness is a precursor to, or a lesser grade of, MTSS (Yates & White 2004, Newman et al 2012).

Imaging is repeatedly demonstrating that MTSS is primarily a problem of bone (Beck 1998, Magnusson et al 2003, Magnusson et al 2001, Batt et al 1998, Moen et al 2009, Gaeta et al 2005, Fredericson et al 1995). Computerised tomography, dexa and magnetic resonance imaging techniques consistently identify marrow oedema, periosteal lifting due to underlying bone exudate, and bony resorption of the posteromedial tibial border in MTSS. While the accuracy of different imaging techniques may be dependent on timing of the imaging in relation to injury onset (Ozgurbuz et al 2011, Moen et al 2012), histology studies confirm the presence of tell-tale bone stress markers in MTSS sufferers (Bhatt et al 2000, Johnell et al 1982).

Risk factors

In clinical practice, multiple risk factors are seen as being linked to MTSS as a result of various research findings or as a product of practitioners' beliefs, experiences, biases, and paradigms. The variables referred to by clinicians and sometimes investigated in studies fall into categories of: range of motion (ROM) and muscle length measures including joints from hip to hallux; static posture of lower limb segments; kinematic analyses of lower limb; muscle strength and endurance; running volumes; anthropometric measures; dietary, hormonal, smoking status; past history of injury; orthotic and shoe use. The majority of papers that have reported investigations of these associations have been case-control or retrospective in design, which raises questions about the attribution of cause and effect.

A very recent systematic review and meta-analysis (Newman et al 2013, forgive the gratuitous reference to my own work) has found that female gender, a previous history of

MTSS, fewer years of running experience, prior orthotic use, increased BMI, an increased navicular drop, and increased external rotation hip ROM in males are all factors significantly associated with an increased risk of developing MTSS in runners. The mechanism by which these risk factors influence the development of MTSS remains unclear. This paper also found soleus and gastroc length, low BMI and foot type were not risk factors for MTSS (Newman et al 2013).

Management

A recent systematic review with meta-analysis reveals the low level of evidence available to date (Winters et al 2013). Eleven RCTs and two non-RCT trials were included, involving a range of interventions from braces, to laser, iontophoresis, phonophoresis, ESWT, periosteal pecking, stretching and strengthening. According to the authors, none of the papers were sufficiently free of bias to confidently rely on the results (level 3–4 evidence). Of all the therapies analysed in the review ESWT shows the most potential as an intervention.

To assist the clinician a summary table (Table 1) is included as a rough guide for treatment selection.

Table 1.

	Treatment ideas	Level of evidence	Therefore
MTSS	Cushion insole/orthotic	1b +ve, 2a -ve	Try and evaluate
	stretch	2a-ve, 1-ve	Probably don't bother
	Strengthening calf	2b-ve,1-ve	Probably don't bother
	Foot intrinsic strength	3+ve, 4+ve	Try it, more research needed

	Taping/bracing	2-ve, 1b-ve	Probably don't bother
	Running technique	3 +ve and 3-ve	More research needed
	Periosteal pecking	4+ve	More research needed
	Muscle release/STM	3+ve, 3-ve	Try it, more research needed
	Balance/control	2-ve, 3 +ve	Try it, more research needed
	Subtalar joint 'function'	Nil ?4+ve	More research needed
	Preconditioning	2-ve, 3 +ve	Makes sense, try it, more research needed
	Graduated running program	2 +ve, 2-ve	Try and evaluate, more research needed
	ESWT	2 +ve	More research needed

1a—Evidence from meta-analysis of randomised controlled trials; 1b—Evidence from at least one randomised controlled trial; 2a—Evidence from at least one well designed controlled trial which is not randomised; 2b—Evidence from at least one well designed experimental trial; 3—Evidence from case, correlation, and comparative studies.; 4—Evidence from a panel of experts

CECS: 14–27 per cent of runners

CECS is characterised by pain on exertion that comes on rapidly with increasing intensity and settles within minutes of rest. Raised intramuscular pressure (IMP) within the fascial compartment impedes local blood flow and affects neuromuscular function of the compartment tissues. In severe cases muscle necrosis can ensue. The pathophysiology of

CECS is unknown. Decreased fascial compliance, increased fascial thickness, rapid muscle hypertrophy with insufficient fascial accommodation, abnormal fluid volume within the compartment, or shorter periods of muscle relaxation have all been postulated—but evidence is lacking to support these theories.

There is some evidence that shorter periods of muscle relaxation, measured by EMG, are a component of the problem (Zhang et al 2011). Measures of fascial thickness and compliance have not detected any differences between normals and CECS sufferers (Dahl et al 2011, Barbour et al 2004). Lower leg muscles have been estimated to increase their volume by up to 20 per cent during intense exercise, but how this may differ between CECS sufferers and normals is unclear (Schissel & Godwin 1999).

CECS is usually diagnosed objectively via needle manometry measurement of IMP. This is considered to be the gold standard, but a growing body of evidence is casting serious doubt on the reliability of needle manometry. Besides being invasive and uncomfortable, the diagnostic criteria currently used to diagnose the condition using needle manometry come from very small sample sizes of symptomatic patients. Studies since have shown that differences in intramuscular pressure occur between males and females, athletes and non athletes, between compartments (anterior to posterior, deep to superficial), and between measurement methods. As a result there is considerable overlap in the ranges of the diagnostic criteria and those found in asymptomatic subjects (Hislop & Tierney 2011, Pedowitz et al 1990, Roberts & Miller 2011).

Further, intramuscular pressures correlate poorly with pain and neural symptoms (Gentilello et al 2001). In other words, the diagnostic methods used for CECS may not be valid. How many studies about this condition have misclassified their participants and thus diluted potential findings?

A new application of old technology is showing exciting promise in this regard. Near infra-red spectroscopy (NIRS) is a non-invasive method of measuring local muscle oxygenation. It is a more direct measure of ischaemia than IMP, and it has been shown to compare favourably to functional MR and needle manometry, but further studies are required to further validate this technique (van den Brand et al 2005, van den Brand et al 2004) (See Figures 1 and 2).

Risk factors

There is a vast gap in the literature investigating the risk factors for CECS. A rapid increase in volume of lower limb exercise may lead to rapid hypertrophy and a consequent vicious cycle of pressure increase. Microvascular and possibly neural damage associated with diabetes may be a risk factor. There may even be genetic factors associated, as found in a case study of twins with the condition (Banerjee & Mclean 2011). There is much more to learn in this area.

Management

Management of CECS is very difficult. ‘Rest is best’, but the patients who get this condition are usually the ones most resistant to resting. The literature on management of CECS tends to include elements of graduated running programs, needling, running technique and kinematic modification, stretch, strength, soft/deep tissue massage and surgery involving fasciotomy/fasciectomy. The latter has had the most reports of success, but with success rates being as low as 60 per cent and serious complications in up to 15 per cent of cases, it should not be a first option unless the condition is severe. Some initial evidence has shown adopting a forefoot strike pattern may be of benefit (Diebal et al 2012), but larger trials are needed that also monitor adverse effects from the intervention. Hypertonic glucose prolotherapy has also been trialled (Lyftogt 2006).

To assist the clinician a summary table (Table 2) is included as a rough guide for treatment selection.

Table 2.

	Treatment ideas	Level of evidence	Therefore
CECS	Running technique	2b +ve and 3-ve	Start here with caution, might depend on compartment(s) involved. More research needed
	Graduated running program	2b +ve, 2b-ve	Start here, more research needed
	Myofascial massage	Nil found	Usually worsens
	Fasciotomy	3+ve	Exhaust other options first, 60–100 % 'success'. More research needed, careful post op routine
	Prolotherapy—thought is to sclerose neovessels?	4 +ve early experimental	More research needed to proceed with any confidence

1a—Evidence from meta-analysis of randomised controlled trials; 1b—Evidence from at least one randomised controlled trial; 2a—Evidence from at least one well designed controlled trial which is not randomised; 2b—Evidence from at least one well designed experimental trial; 3—Evidence from case, correlation, and comparative studies.; 4—Evidence from a panel of experts.

PAES: 0.2-3.5 per cent

Clinicians are becoming much more aware of PAES as an important differential diagnosis in recalcitrant cases of CECS, but it remains an illusive diagnosis for many

patients (Politano et al 2012). The popliteal artery can be entrapped by muscle or fascia or space occupying lesions, producing claudicant like symptoms very similar to CECS (Gourgiotis et al 2008). It is relatively easily diagnosed by arthrogram or Doppler ultrasound (Nelms et al 2000); however, depending upon the nature of the entrapment it may be important to perform these tests in functional positions (calf raise) or post exercise to ensure an accurate diagnosis (Macedo et al 2003, Wright et al 2004).

Risk factors

PAES is reported to occur in men more than women at a rate of 15:1, although these figures come from studies in military populations that are likely to be skewed (Bouhoutsos & Daskalakis 1981). Larger calf musculature, rapid hypertrophy, athletic frame and genetics have also been implicated (Gourgiotis et al 2008). Essentially anything that has the potential to obstruct, entrap or constrict the popliteal vessels is a risk for the condition.

Management

This is a problem of the anatomy. The best way to fix it is to get the anatomy fixed by someone who knows what they are doing: a vascular surgeon. The procedure is not without its risks, so due consideration must be given to the severity of the impairment, the nature of the entrapment, and the likelihood of resolution.

Tibial Stress Fracture: 1.5–31 per cent of runners

Readers will be aware of the significance and prevalence of stress fractures in the sporting population. Typically, patients will present with very localised, intense pain to palpation or vibration. They may report pain at rest, pain at night, and certainly pain with load or impact. Plain X-ray won't necessarily detect a stress fracture for some time, so these are best diagnosed by CT, MR or triple phase bone scan. The tibia is the most common stress fracture site in athletes (Milner et al 2006, Matheson et al 1987). This is

an important differential diagnosis for a patient presenting with CELP, as the management principles are as clear as the consequences of misdiagnosis.

Risk factors

Stress fractures are essentially a problem of overload: too much too soon. Technique and kinematic factors related to footstrike pattern, excessive valgus, ‘free moment’ or torsion in stance phase have been identified as potential risks. Genetics, bone geometry, gender, hormonal status, metabolic and nutritional factors have also been suggested to play a part in the development of bone stress (Milner et al 2006, Milgrom et al 1985, Milner et al 2011).

Management

Some stress fractures are more unstable than others, usually determined by their extent and location. Grading the fracture will determine how long to rest, whether to immobilise and how much to load bear. Patient education, technique modification and addressing any other risk factors would be important prior to return to activity.

Discrete syndromes or a continuum?

Traditional views of CELP syndromes consider each diagnostic entity as separate pathologies that can sometimes coexist (Figure 3). Some authors have developed grading systems based around this model of understanding (Detmer 1986). The fact that these conditions can co-exist is well documented, but not well understood. What this model fails to do is to consider likely pathways or precursors that may put an individual at risk of each pathology. In order to consider how these conditions may interact or predispose to each other, it may be more useful to consider a continuum type model (Figure 4). Readers interested in a practical approach to differential diagnosis of these conditions are recommended to refer to Edwards et al (2005).

Further research is sorely needed to better understand why these conditions develop and how they interact. Growth in this understanding should allow us to be more definitive in identifying risk, more informed in injury prevention, more confident in diagnosis, more accurate in prognosis, and more evidence based in our prescriptions.

For full references, email ngeditor@physiotherapy.asn.au.

Figure 3. Traditional model of CELP differential diagnosis.

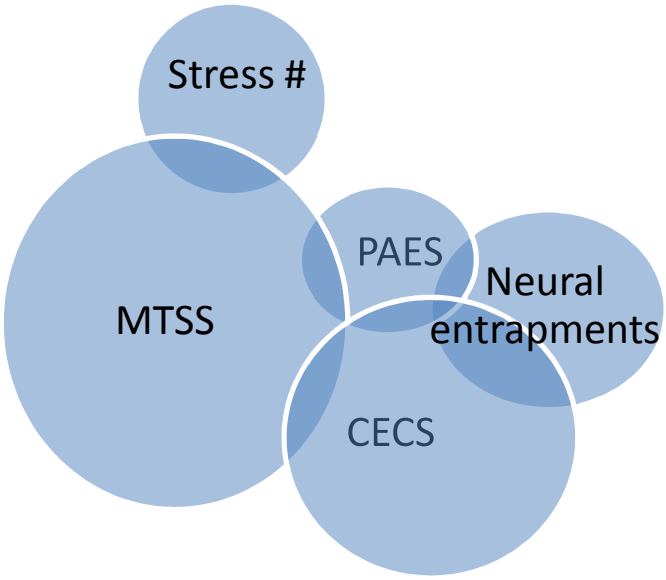
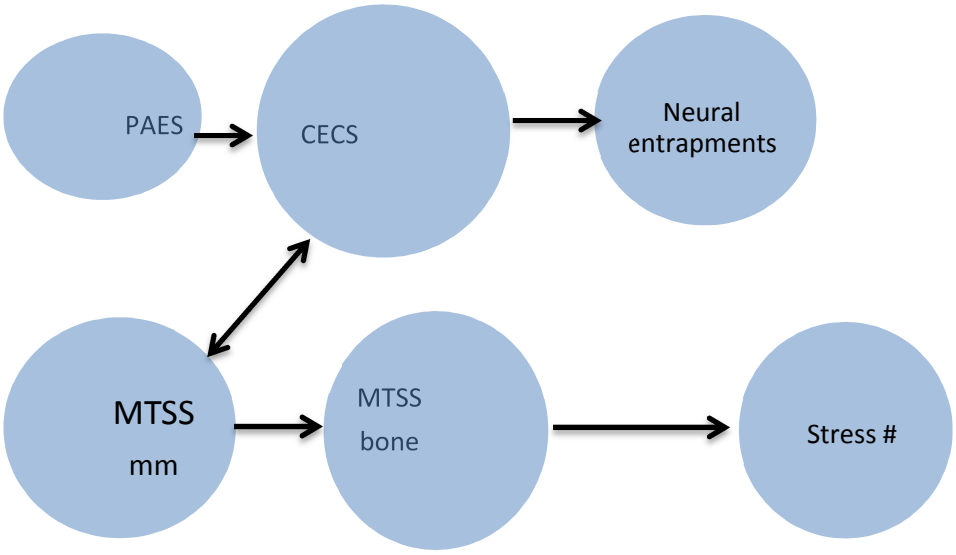


Figure 4. Proposed continuum model of CELP differential diagnosis.



	Channel 1	Channel 2	Channel 3	Channel 4
Reference Level (%):	50.00	50.00	50.00	50.00
Baseline Regional Oxygen Saturation (%):	60.18	62.16	-	-
Session End Regional Oxygen Saturation (%):	60.78	64.22	-	-
Cumulative Saturation Below Threshold (min-%):	3	0	0	0
Baseline from Oximeter (%):	69.00	67.00	-	-
Oximeter Provided First Indication:	No			

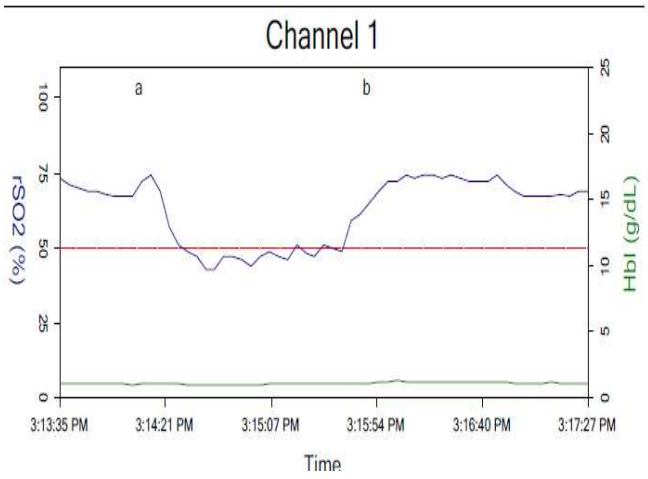


Figure 1 NIRS trace. Regional oxygen saturation measured over tibialis anterior in an asymptomatic runner a=onset of running b=cessation of running

	Channel 1	Channel 2	Channel 3	Channel 4
Reference Level (%):	50.00	50.00	50.00	50.00
Baseline Regional Oxygen Saturation (%):	65.18	69.91	-	-
Session End Regional Oxygen Saturation (%):	87.02	80.18	-	-
Cumulative Saturation Below Threshold (min-%):	27	0	0	0
Baseline from Oximeter (%):	65.00	69.00	-	-
Oximeter Provided First Indication:	No			

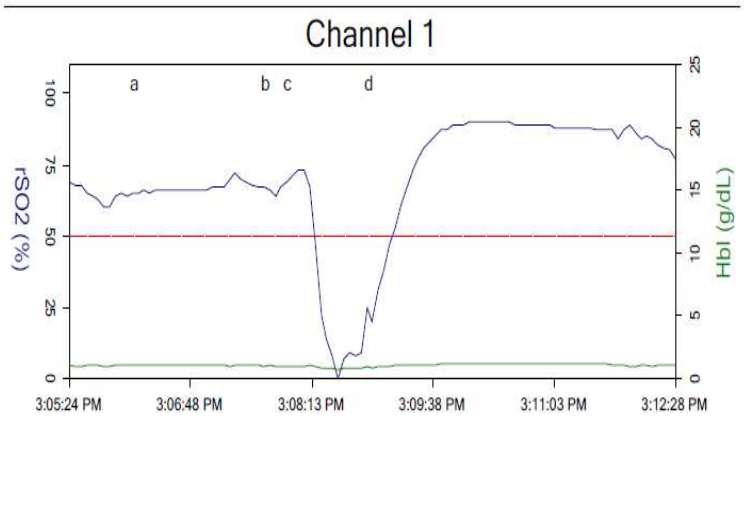
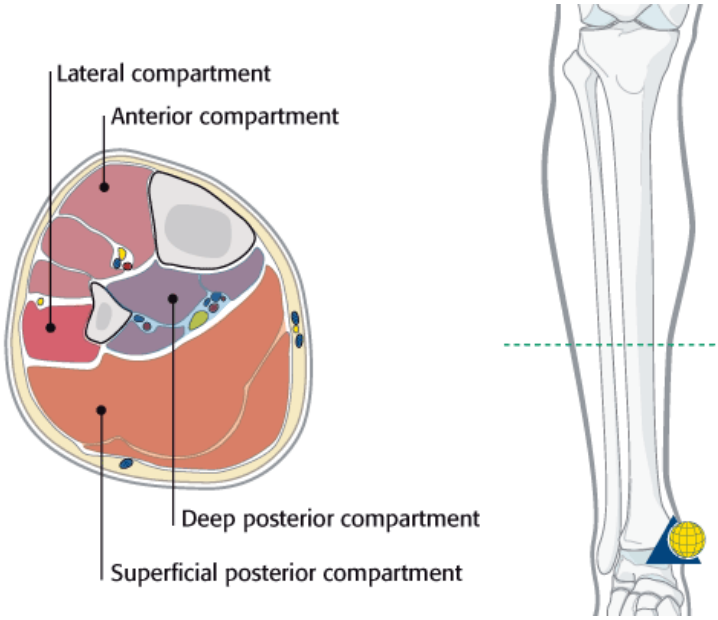


Figure 2 NIRS trace. Regional oxygen saturation measured over tibialis anterior in a CECS patient b=onset of running d=cessation of running



Fascial compartments of the lower leg

aofoundation.org (last accessed 18/2/13)